

Estimating the risk of percutaneous coronary intervention

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Abstract

Percutaneous coronary intervention (PCI) is expanding in terms of both the numbers of patients treated and the scope and severity of coronary artery disease tackled. These developments have occurred in parallel with increased awareness of the importance of accountability and clinical governance. Whilst cardiac surgeons have durable risk scores such as Parsonnet and EuroSCORE to assist them and their patients with estimating procedure-related risks, interventionists lack such universally accepted tools. Or do they? In this paper, we review the available PCI risk scores and point out the pressing need for the systematic use of a robust, simple and widely acceptable risk score for routine clinical use.

Key words: percutaneous coronary intervention, risk score.

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Introduction

Since Gruentzig performed the first coronary angioplasty nearly 30 years ago,¹ this field has seen rapid expansion. From a technique initially confined to the treatment of simple, short lesions in large, proximal vessels, it has evolved to one that is now used to treat a wide range of complex lesions. The patient profile is also changing. In the early days, generally only patients with chronic stable angina were treated. The role of percutaneous coronary intervention (PCI) has now broadened, following the expanding evidence base for its use in unstable angina, non-ST elevation myocardial infarction (NSTEMI), ST elevation myocardial infarction (MI) and cardiogenic shock.²⁻⁵

In parallel with expansion of capability has come increased accountability. The principles of clinical governance, coupled with the prevalent political imperative of transparency, dictate

that interventionists' results should be available and they should be on a par with those of their peers. Patients, the government and the media have also embraced this philosophy. The publishing of raw mortality data for individual operators is imminent, probably with little or no allowance for 'case mix'. Part of the reason for this lies within the profession itself, in that there is no simple, universally accepted risk score for patients undergoing PCI. Cardiothoracic surgeons use risk prediction models, such as Parsonnet and EuroSCORE, on a daily basis in clinical practice.^{6,7} These scoring systems, albeit imperfectly, attempt prediction of death and serious complications in patients being considered for cardiac surgery on the basis of some fairly simple risk factors. They allow both surgeon and patient to be informed about peri-operative risk. For PCI, in contrast, procedural risk is either quoted as an overall national, institutional or personal figure, or, if the person is deemed at high risk, this risk is modified upwards on an *ad hoc* and subjective basis. As the scope of interventional cardiology broadens and the complexity of cases increases, there is a pressing need to quantify individual risk and to alert both the patient (who is increasingly becoming well informed) and the cardiologist to the likelihood of an adverse outcome.

This article will review the risk scoring models for PCI which are currently available. We will then draw some general and practical conclusions and point the way towards a widely acceptable scoring system. We will discuss the major risks of the procedure itself (notably mortality, but also MI, stroke and emergency coronary artery bypass grafts [CABG]) and not the medium-term (and less threatening) risk of restenosis.

The RSCAI model

Kimmel *et al.* published a simple additive scoring system for predicting death, MI and emergency CABG after PCI in 1995 from data collected in 1992 and validated in 1993.⁸ They identified seven variables (table 1) from multivariate, stepwise regression analysis of multiple risk factors in 10,622 patients who underwent angioplasty and were entered on to the Registry of the Society for Cardiac Angiography and Interventions (RSCAI) in the United States. The authors added the number of risk factors present and classified patients into four groups, depending on the number of risk factors they possessed. Those with no risk factors were classified as low-risk (complication rate 1.3%); those with one or two risk factors as moderate-risk (complication rate 2.8%); those with three risk factors as high-risk (complication rate 12.7%); and those with four or more risk factors as very high-risk (complication rate 29.7%). The authors tested their model on patient data from

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Table 1. Variables used in PCI scoring systems

	RSCAI	NNE	INTERVENT	Michigan	Boston	King's	Mayo	Michigan-II	New York
First author	Kimmel	O'Connor	Budde	Mosucci	Resnic	de Belder	Singh	Qureshi	Wu
Era of study	1992	1994–96	1996	1997–99	1997–99	1995–97	1996–2001	1996–98	2002–03
No. of patients	10,622	15,331	455	10,796	2,804	1,500	5,463	9,954	46,090
Recent acute MI	Y	Y	Y	Y	Y			Y	Y
Age		Y	Y	Y	Y	Y	Y	Y	Y
Cardiogenic shock	Y	Y		Y	Y	Y	Y		Y
Urgent/emergency PCI		Y			Y		Y		
Female sex			Y	Y					Y
Haemodynamic instability									Y
LVEF		Y		Y		Y			Y
CCF		Y	Y		Y		Y		Y
IABP <i>in situ</i>		Y							
Previous MI									Y
Prior cardiac arrest				Y					
PVD		Y		Y					
Renal failure		Y		Y	Y	Y	Y	Y	Y
Prior thrombolysis						Y			
Unstable angina	Y		Y		Y				
Left main angioplasty	Y				Y		Y		Y
Multi-vessel disease	Y		Y	Y			Y	Y	
Thrombus at lesion site				Y			Y		
AHA lesion type	Y	Y	Y	Y	Y				
No stent deployed					Y				
Aortic valve disease	Y								

Key: MI = myocardial infarction; PCI = percutaneous coronary intervention; LVEF = left ventricular ejection fraction; CCF = congestive cardiac failure; IABP = intra-aortic balloon pump; CABG = coronary artery bypass grafts; PVD = peripheral vascular disease; RSCAI = Registry of the Society for Cardiac Angiography and Interventions; NNE = Northern New England; AHA = American Heart Association; Y = yes

the following year and found the reproducibility of the index to be excellent, $p < 0.00001$.

This scoring system was a pioneering achievement, but it had a number of limitations. First, the risk factors were all regarded as having equal importance and therefore all received the same weighting. This is now thought to be incorrect, because certain risk factors, such as cardiogenic shock and severe renal disease, are recognised as particularly powerful predictors of adverse events. Second, the authors restricted the model to only seven risk factors, potentially limiting the accuracy and validity of the model. Third, the model was based upon data from a rather distant era, prior to the routine application of PCI in multivessel disease and acute coronary syndrome, stenting and anti-platelet agents.

Northern New England model

O'Connor *et al.* produced a Northern New England (NNE) model based upon data collected from 15,331 patients who underwent PCI between 1994 and 1996 (table 1).⁹ These data were used to develop and internally validate a multivariate prediction equation

for in-hospital mortality. A goodness-of-fit (Hosmer-Lemeshow) test was not significant (indicating that the model departed little from a perfect fit) and the receiver operating curve (ROC: a statistical method of determining model accuracy; a value of 1 indicating a perfect model, and a value of 0.50 a model whose predictive ability is no better than 50–50) for the probability of death was 0.88, indicating a satisfactory ability to identify patients at high risk.

Holmes *et al.* tested this model on the National Heart, Lung and Blood Institute (NHLBI) registry of patients (1997–99) and found that it predicted in-hospital mortality in 1.3% of patients (95% confidence intervals [CI] 1.0–1.7%), compared with an observed mortality of 1.4%.¹⁰ This suggested that the model was still effective in the stent and (emerging) glycoprotein IIb/IIIa inhibitor era. Kizer *et al.* also tested the NNE model on patients from the stent and adjunctive pharmacotherapy era, and found good discrimination but poor goodness-of-fit. In Kizer's hands, the model significantly overestimated mortality (predicted mortality 1.1%; observed mortality 0.4%).¹¹ There remains significant doubt, therefore, as to whether the NNE model, and other

models devised a decade ago, accurately predict current outcomes.

The INTERVENT project

A European group collaborated on the INTERVENT project with the purpose of setting up a prognostic, computerised model to predict individual outcome after PCI.¹² Data obtained from 455 patients undergoing PCI were analysed by machine learning, a method derived from techniques used in artificial intelligence. Nine risk factors were found to be associated with death or MI after a PCI (table 1). The INTERVENT computer programme stratified patients into three categories: low-risk (probability of an adverse event < 10%); moderate-risk (probability 70–90%); and high-risk (90–95%). This model had a 97% accuracy for predicting the risk of an adverse event following a PCI, but its value as a tool to clinicians and patients is limited by the risk banding, which is unrepresentative of both the doctor's and the patient's understanding of low, moderate and high risk. The model was also based upon a small dataset of 455 patients and may have misrepresented the importance of various risk factors. Furthermore, the rate of stent usage was less than 25% among the dataset analysed.

The Michigan model

The Michigan model recorded patient demographics, co-morbidity and lesion characteristics from 10,796 patients who underwent angioplasty between 1997 and 1999 in eight US hospitals.¹³ Logistic regression analysis was performed to identify predictors of in-hospital death (table 1). The score was validated on a second group of 5,863 patients who underwent angioplasty between 1999 and 2000. The authors reported near perfect correlation between scores and mortality rates. In the low-risk group (score < 1.5), the mortality rate was 0.05%, whereas in the high-risk group (score > 5), the observed mortality was 34.3%. The area under the ROC was 0.91, consistent with excellent model discrimination.

The Michigan model yielded a plot of risk versus mortality. The clinician calculates an individual's risk score, enters it onto the x-axis and reads the predicted mortality from the y-axis. The curve is sigmoidal in shape. Risk remains low below a score of 3 and then increases rapidly until a score of 8 corresponds to an in-hospital mortality of 64%. This simple system is a useful tool for predicting risk, but has been validated only for in-hospital death and not for all major adverse cardiac events (MACE). It does, however, reveal just how low the mortality rate is when the risk score is low. Holmes *et al.* applied this model to patients who underwent PCI between 1997 and 1999 in the NHLBI dynamic registry.¹⁰ Observed in-hospital mortality rate was 1.4% and the predicted rate was 1.1% (95% CI 0.7–1.3%).

The Boston model

Resnic *et al.* compared the capabilities of three types of risk score: a simple integer score, one with full logistic regression and one utilising a computerised neural network.¹⁴ The authors developed their models from 2,804 consecutive cases between 1997

and 1999 at the Brigham and Women's Hospital, Boston, and then retrospectively validated the models with data from 1999. They identified 10 risk factors (table 1) for adverse outcome and one protective factor against adverse outcome (coronary stenting) by multiple logistic regression. The authors tested the models for their ability to predict in-hospital death and the composite of in-hospital death, acute MI and emergency CABG. The ROCs of all three models were almost identical. The areas under the curves were calculated (for mortality and the composite end point, respectively) as 0.86 and 0.74 for the simple integer model, 0.85 and 0.78 for the logistic regression model, and 0.83 and 0.81 for the neural network model. The Hosmer-Lemeshow goodness-of-fit test was adequate for all the models except the simple integer score, which was non-significant in the composite end point group.

The authors concluded that the simple integer risk score performed as well as the more complex logistic regression model and neural network models. Limitations of this model included the changing usage of glycoprotein IIb/IIIa inhibitors during the study, from 36% in the development dataset to 53% in the validation set, the lack of external validation and retrospective analysis.

The King's College Hospital model

De Belder *et al.* developed a model to predict MACE after PTCA by utilising Bayes' Theorem.¹⁵ This states that future outcomes can be predicted from known outcomes. As applied by the King's group, their model predicted the probability of an adverse outcome based upon the patients' known risk factors. These risk factors were derived from the construction of a Bayes table of 26 patient-derived variables based upon 1,500 procedures performed between 1995 and 1996. The authors tested the model on 1,000 patients who underwent PTCA between 1997 and 1998. They found that the area under the ROC was 0.76, a moderate predictive power. The current applicability of this study is limited by the smaller numbers involved compared to the US studies and by the changing practice of PCI. The stent rate in the development group was 54% and use of abciximab was 0.5%, rising to 5.1% in the evaluation group. The identification of only five risk factors (table 1), whilst conferring simplicity, limits the discriminatory power of the model and may reflect the relatively small numbers of patients entered into the evaluation group. Nevertheless, practicality, simplicity and applicability to British practice make this model attractive.

The Mayo Clinic model

Singh *et al.* developed a simple, integer risk score from 5,463 patients who underwent PCI between 1996 and 1999 at the Mayo Clinic.¹⁶ This period coincided with the widespread utilisation of stents (82% in the study group), thienopyridines (90%) and glycoprotein IIb/IIIa inhibitors (42%), reflecting current practice more closely than other models. Logistic regression analysis identified five clinical and three angiographic variables which correlated with MACE (table 1). These were then assigned integer values proportional to the estimated continuous coefficients

from a logistic model. Patients were typically found to score between 0 and 25. The authors divided this range into five categories: score < 5 (very low-risk); 6–8 (low-risk); 9–11 (moderate-risk); 12–14 (high-risk); and ≥ 15 (very high-risk). The observed (vs. predicted in parentheses) complication rates in the validation set for these groups were, respectively: 1.0% (< 2%), 3.0% (2–5%), 6.2% (5–10%), 19.5% (10–25%), and 35% (> 25%). The authors reported an area under the ROC of 0.76 and the Hosmer-Lemeshow test revealed that the model fitted the data well. The model predicted the risk of the various groups well. Individual patient risk could be estimated from a plot of score versus risk. The study proved the validity of a simple additive integer score, based on relatively few simple variables. The main limitation was that the data were derived from a single centre without external validation. The authors also excluded from the study certain patients, such as those who had undergone previous PCI.

The authors have, since, validated the Mayo Clinic risk score in an independent data set of 3,264 patients from 17 sites.¹⁷ There were 96 (2.94%) observed complications and the score predicted 93.5 events. The Hosmer-Lemeshow goodness-of-fit p value was 0.28 and the area under the ROC curve was 0.76. This model seems to be robust, relatively simple and recent by comparison with the others.

The Michigan-II model

Qureshi *et al.* devised a simplified scoring system for predicting mortality after PCI.¹⁸ Demographic, clinical and procedural data were recorded on 9,954 patients who underwent PCI between 1996 and 1998 in a single centre. Data were prospectively collected on age, sex, coronary risk factors, renal function, extent of coronary artery disease, recent MI, history of CABG and history of peripheral vascular disease. Patients undergoing PCI between June 1999 and November 2001 were studied as a validation group. In-hospital mortality was the primary end point. Crude mortality and univariate odds ratio of mortality for different clinical factors were calculated. Individual risk factors that were significant in the univariate analysis were entered into a stepwise, multiple, logistic regression model to determine the most parsimonious model that predicted mortality.

Four factors that had the highest impact on mortality (recent MI, multi-vessel disease, elevated creatinine and age > 65 years) were used for further analysis and the development of a risk score. These factors were given individual weighting at univariate analysis. The total risk score for an individual determined assignment to one of four classes. Crude mortality of the individual classes in the derivation (and validation) groups was respectively: class 1, 0.1% (0.1%); class 2, 0.5% (0.4%); class 3, 2.3% (2.3%); and class 4, 9.1% (7.1%). In the validation subset, the area under the ROC curve was 0.83. This was a large study and it provided outcome evaluation of a reasonably contemporary interventional population. The authors did not comment, however, on the proportion of patients receiving stents and glycoprotein IIb/IIIa inhibitors. This risk score used only four risk factors, which may underestimate the influence of other potentially

Table 2. Five hypothetical examples of patients undergoing PCI to test each scoring system

Case 1	52-year-old male Elective admission for stable angina pectoris No co-morbid factors Good LVEF Mid-LAD stenosis; simple anatomy
Case 2	67-year-old female Elective admission for stable angina pectoris Type 2 diabetes, hypertension A previous MI two years ago Good LVEF LMS stenosis; mid-LAD stenosis
Case 3	58-year-old male Acute presentation with chest pain at rest ECG: ST elevation anterior leads No co-morbid factors Haemodynamically stable Triple vessel disease; thrombus mid-LAD
Case 4	81-year-old female Admitted with NSTEMI two days ago Oral aspirin and nitrate infusions Diabetes, previous inferior MI LVEF 40% TIA six weeks ago Double vessel disease. Occluded RCA, diffuse, long, calcified circumflex stenosis
Case 5	73-year-old female MI 16 hours ago Diabetes, hypertension, moderate COPD SBP 86 mmHg, pulse 104 bpm, oliguric Creatinine 228 $\mu\text{mol/L}$ LVEF < 30% Subtotally occluded LAD

Key: LVEF = left ventricular ejection fraction; LAD = left anterior descending artery; MI = myocardial infarction; LMS = left main stem; NSTEMI = non ST-elevation myocardial infarction; TIA = transient ischaemic attack; RCA = right coronary artery; COPD = chronic obstructive pulmonary disease; SBP = systolic blood pressure; PCI = percutaneous coronary intervention

important factors like left ventricular function and lesion complexity. The score predicted mortality only, rather than MACE.

The New York model

Very recently, Wu *et al.* have published the New York risk score to predict in-hospital mortality following PCI.¹⁹ The risk score was derived from 46,090 patients who underwent PCI in 41 different hospitals across New York state in 2002 and then validated on 50,046 PCI patients who underwent PCI in the same hospitals in 2003. The data were obtained from a registry (The New York State Percutaneous Coronary Intervention Reporting System) that collects detailed information on each patient's demographic characteristics, pre-procedural risk factors, complications and discharge status.

A logistic regression model was developed to predict in-hospital mortality using a cross-validation strategy. From this, a risk score was derived. This included nine risk factors: age, sex,

Table 3. Predicted PCI risks (in percentage) of the five hypothetical cases as calculated by each scoring system

	RSCAI	Michigan	Boston	Mayo	Michigan-II	New York	EuroSCORE	Parsonnet
Case 1	1.3	< 0.8	0.4	< 2.0	0.2	0.05	0	2
Case 2	2.8	< 0.8	0.4	8–9	0.5	0.81	3	9
Case 3	2.8	< 2.0	2.7	5	2.3	0.59	4	12
Case 4	12.7	4	16.7	10	0.5	1.12	11	28
Case 5	2.8	> 50	21.4	> 25	9.1	49.01	17	34–84*

A head-to-head comparison of the six systems that can be easily applied at the bedside. EuroSCORE and Parsonnet scores are also shown. The systems test the five hypothetical cases shown in table 2.* Predicted risk varies depending upon the perceived severity of cardiogenic shock. The Parsonnet score allows 10–50 points for 'catastrophic state'

haemodynamic state, ejection fraction, recent myocardial infarction, peripheral arterial disease, congestive heart failure, renal failure and left main disease. The 'weighting' of each risk factor varied from 1 to 9 and the total risk score from 0 to 40. The predicted risk of in-hospital death varied from 0.05% for a score of 0, to 21% for a score of 20, to 99.36% for a score of 40. Only 0.5% of patients had a score > 19 and no patient had a score > 31 (risk of 90%). When applied to the validation subset of 50,046 patients, the score predicted a mortality of 0.66% when the actual mortality was 0.58%. The discrimination of the model was very high ($C = 0.905$) when applied to the 2003 dataset. This risk score is based on a huge dataset taken from the contemporary PCI population. It is simple to use and appears to be reliable. It does not include the urgency of the procedure or any lesion-related variables. Also, this score predicts in-hospital mortality only and not MACE.

Some examples

It may come as a surprise to the reader that there are so many apparently well-developed risk-scoring systems for PCI available in the literature. But do they work? To test how these different systems would function in practice, we applied them to five hypothetical cases (table 2). These cases are intended to embrace a range of severity of cardiac, co-morbid and lesion-related conditions that may be encountered by an interventional cardiologist in day-to-day practice. We then calculated the PCI risks of these cases on the basis of the six of the nine risk scores reviewed above which were easy to calculate at the bedside without the need of a calculator or a complex computer programme. We also calculated their EuroSCORE and Parsonnet scores to place their estimated PCI risk into context. The results are shown in table 3.

A number of points emerged from this exercise. For the apparently (that is, intuitively) low-risk cases, all the systems predicted risk scores that were not very different from one another. The only exception to this was the Mayo Clinic score, which predicted a higher risk of 8–9% for Case 2. It may be important to note here that, unlike many other scores that predicted in-hospital mortality only, the Mayo Clinic score was designed to predict MACE. This score gives high weighting to LMS disease, multi-vessel angioplasty and increasing age and all this may have

resulted in Case 2 having a higher predicted risk using this scoring system.

In the apparently (intuitively) high-risk cases, though, the risk scores varied in their assessments quite markedly. Noticeable among this group was a prediction of low risk for Case 4 by Michigan-II and New York and for Case 5 by RSCAI. The Michigan-II model included only four variables in its scoring system, excluding such potentially significant predictors as left ventricular (LV) function, emergency procedure and lesion characteristics. The New York model did not include variables such as lesion characteristics and urgency of the procedure (with the exception of acute STEMI) and this may have led to an under-estimation of risk. The RSCAI model did not include many known predictors of PCI risk, including age, renal failure and emergency procedure, whilst assigning equal weight to all risk factors. Taken together, these factors may have led to an under-estimation of risk by these models.

As far as the surgical scores are concerned, both EuroSCORE and Parsonnet showed wide discrimination between the cases in our exercise. Parsonnet scores were higher than those from EuroSCORE for each of the cases. It is interesting to note that, for Case 5, Parsonnet could allocate any score between 10 and 50, depending upon the perceived severity of the cardiogenic shock, explaining why this system provided a risk that lay between 34 and 84%. Parsonnet appears to allow a large subjective element into an otherwise objective system.

Overall, the models tested were simple to use. The Michigan-II model was the easiest, with only four variables, probably at the cost of poor discrimination. The Boston and Mayo Clinic models showed wider discrimination than other models. It is impossible to say which is the most accurate, but it is hard to argue against the sheer numerical power of the New York system.

Summary

To summarise the findings of the studies reviewed here, the main risk factors for MACE after PCI are age, recent MI, cardiogenic shock, emergency PCI, female sex, poor LV function, renal impairment, peripheral vascular disease, multi-vessel disease, left main angioplasty and American Heart Association/American College of Cardiology (AHA/ACC) type C lesion. A simple, aver-

age figure for the risk of adverse outcome, quoted to all patients, would over-estimate the risk for a large number of patients and grossly under-estimate the risk for a few. For low-risk patients, this practice may cause undue anxiety and, for high-risk patients, undue reassurance.

Risk scoring: why don't we do it?

Notwithstanding their limitations, this review has described several well-developed PCI risk scoring systems, which have been published over the last decade and a half. Why, then, are they not generally used? It is clear from this review, and especially from the 'head-to-head' test, that several of the systems have marked deficiencies. First, some are overly complex and computer-based, whereas simple, additive, integer models are preferable in a busy practice. Second, some reflect outdated practice. Recent studies by Holmes and Kizer questioned the validity of these studies to current patient outcome.^{10,11} Kizer, in particular, demonstrated a significant over-estimation of risk in these models. Third, some lack credibility, chiefly relating to the number of patients studied (which range from 455 to more than 100,000) and the number of risk factors included (four to 13). Many of the studies were also limited to data derived from a single centre, limiting their applicability to other centres or countries. Fourth, only a few of the studies use total MACE as their end point, which is inadequate, because stroke, MI and emergency CABG are important outcomes. Fifth, the commonly used end point in these studies was 'in-hospital' death or other MACE, and this might not fully reflect events in the first 30 days (particularly in the context of PCI for acute coronary syndromes [ACS], which now accounts for up to 50% of cases). A 30-day end point would also be more symmetrical with the surgical scoring systems.

A special problem of predicting PCI risks is posed by the definition of MI. This problem arises from the measurement of troponin release after PCI. Procedural MI is difficult to define and is lacking a consistent policy of action. On the one hand, a joint ESC/ACC document on the definition of MI has suggested that any rise in troponin following PCI should be diagnosed as an MI.²⁰ On the other, a rise in troponin occurs following more than a third of all PCIs,²¹ and in the vast majority of cases is not associated with any long-term problems. It seems excessive to tell patients that they have a greater than 30% risk of having a 'heart attack' as a result of a procedure. Currently, not all patients have cardiac markers measured after PCI and many are treated as a day case, where such measurement is difficult to arrange. None of the risk scores described in this paper used troponin measurement. It may be appropriate to use the old World Health Organisation (WHO) criteria to diagnose a periprocedural MI (the presence of two of: classical symptoms, rise in cardiac enzyme of > 2 or 3 times the upper limit of normal, and ECG changes).

Perhaps the most potent reason for the lack of uptake of risk scoring for PCI, up to now, has been the lack of pressure to do so. Here, the contrast with surgical practice is clear. Surgeons are used to the concept of having outcome data available for peer



Key messages

- The main risk factors for major adverse cardiac events after percutaneous coronary intervention (PCI) are:
 - Age
 - Recent myocardial infarction
 - Cardiogenic shock
 - Emergency PCI
 - Poor left ventricular function
 - Renal impairment
 - Multi-vessel disease
 - Left main angioplasty
 - American Heart Association/American College of Cardiology (AHA/ACC) lesion type

review and (more recently in the UK) publication in national newspapers. The positive benefit of this has been the widespread understanding of what constitutes high surgical risk. There has, however, been a consequent reluctance by surgeons to take on high-risk cases for fear of 'spoiling' non-risk-adjusted outcome data. There is an inevitable move in the same direction in interventional cardiology, and if we fail to have (and use) a risk score, there will be no possibility of risk adjustment in the future.

What we really need

There is a need for an up-to-date, simple, practical scoring system based upon a study which records a complete set of patient risk factors, identifies accurately those that are significant, is prospective, uses data from a number of centres and which uses 30-day major adverse cardiac events as its end point. It would need to be validated prospectively. The risk prediction it produces needs to be readily comprehensible to the clinician and patient in a straightforward and meaningful way. This programme should be based upon nationally collected statistics and be regularly updated by ongoing experience and development of new techniques. For the UK, the Central Cardiac Audit Database (CCAD) project, which will collect data from all centres performing PCI, may be the best way forward in developing such a risk score. While we are waiting, for sheer statistical power and since it is the most recent, we should perhaps adopt the New York score.

Conflict of interest

None declared.

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